Identification of Emerging Subpopulations Susceptible to Adverse Health Effects Associated with Air Particulate Exposure

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The overall weight of evidence from panel, clinical, and toxicological studies has demonstrated the ability of ambient air particulate matter (PM) exposure to induce a variety of extra-pulmonary effects ranging from alterations in hematological parameters to cardiac function. Although a number of mechanisms have been postulated for PM systemic health effects, the causal PM properties and pathophysiological mechanism(s) remain unknown. Toxicological studies have shown the systemic distribution of PM-associated constituents and translocation of particles to other organs following their pulmonary deposition. These toxicological findings raise the following scientific questions: (1) Do additional susceptible subpopulations exist based on these toxicological findings? (2) What are the sensitivity factors, or effect modifiers, within newly identified PM-susceptible subpopulations? (3) What are the PM properties and mechanism(s) of injury responsible for adverse health effects within newly identified sensitive subpopulations? Epidemiology studies have shown diabetics to be twice as susceptible to PM-associated cardiovascular morbidity due to PM's effect on their compromised vascular system. Toxicological studies have provided coherence with this epidemiological association by demonstrating enhanced vasoconstriction in aortas recovered from diabetic rats and altered coronary vasculature function by bioavailable constituents of particulate air pollution particles. Exposure history, disease status, and the nitric oxide synthetase system are sensitivity factors of PM-induced vascular effects observed in aortas from diabetic rats. Subsequent toxicological studies have identified neurotoxic effects in humans and animals following exposure to high air pollution and PM, respectively. A new PM-susceptible subpopulation has been identified by recent epidemiological studies by associating elevated levels of ambient air PM with premature births, birth defects, and low birth weights.

Future Directions: Integrated epidemiological, clinical, and toxicological research efforts are needed to (1) ensure that susceptible subpopulations are identified and characterized with respect to the adverse health effects associated with short- and long-term PM exposure, (2) identify PM properties responsible for adverse health effects within newly identified susceptible subpopulations to link health effects to sources, and (3) determine the mechanism(s) and dose–response relationships associated with adverse health effects observed in newly identified PM-susceptible subpopulations.

Impacts and Outcomes: This research will identify additional PM-susceptible subpopulations as well as link sources to adverse PM health effects within these groups. This information is critically needed by the Agency in order to (1) set PM air standards based on sound science that will protect the most sensitive subpopulations, as mandated by the Clean Air Act; (2) provide information to the Air Quality Index (AQI) public information and notification system; and (3) implement control strategies that decrease levels of causal PM sources. There are potentially 228 million susceptible individuals with health conditions associated with an economic cost of $769 billion that may be adversely affected by PM exposure. Therefore, this research will have a very significant cost-benefit impact by determining the extent to which PM exposure affects these potential susceptible subpopulations.

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